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Case report

Progression of occluded internal carotid artery dissection to giant compressive pseudoaneurysm following gunshot wound

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ABSTRACT

Pseudoaneurysm formation following gunshot wounds is associated with significant morbidity. The natural history and optimal follow-up strategy for penetrating injuries causing dissection remains poorly understood. We report a case of a giant compressive pseudoaneurysm that developed following recanalization of an occlusive internal carotid artery dissection sustained from a gunshot wound. This was detected on routine follow-up imaging six weeks later. The patient subacutely developed dysphagia and hoarseness initially felt to be caused by delayed injury to the laryngeal nerve because computed tomography angiography demonstrated no new pathology. The pseudoaneurysm was successfully treated using a covered stent graft. This case highlights the importance of close angiographic follow-up even in the setting of initial complete vessel occlusion, and the need for a high suspicion for pseudoaneurysm development in the setting of new compressive or neurologic symptoms in patients with potential vascular injury from gunshot wounds to the neck.

1. Introduction

Gunshot injuries to the head and neck are associated with significant morbidity and mortality. In particular, penetrating injury to the neck from gunshot wounds is associated with dissection and pseudoaneurysm formation of the cervical arteries, which can be life-threatening.[1,2] Injury to the internal carotid artery (ICA) is associated with a mortality of up to 18.4% from both immediate and long-term neurological sequelae.[9] The pathophysiology of carotid artery dissection (CAD) involves stenosis or obstruction of the true lumen in subintimal dissection, pseudoaneurysm formation in subadventitial dissection, or intramedial haematoma in cases where blood is restricted to the media. As pseudoaneurysm walls do not contain all layers of the vessel, they are weak and prone to rupture.

The natural history of spontaneous dissection, and (less so) blunt traumatic dissection, has been well studied in the literature.[5] However, the literature regarding penetrating injury, and specifically gunshot wounds involving the extracranial ICA is limited primarily to observational studies in military patients.[4,13] Uncertainty persists as to the best follow-up and management of the patient presenting with dissection from penetrating injury, especially as technical developments have made it possible to reconstruct dissections and restore flow.

We report here on a 26-year-old male who sustained a gunshot wound to the neck and suffered a completely occlusive dissection of the extracranial internal carotid artery that spontaneously recanalized and subsequently developed into a giant pseudoaneurysm causing compressive symptoms. The pseudoaneurysm was managed successfully with an endovascular stent graft. This case illustrates the importance of close angiographic and clinical follow-up in patients with penetrating gunshot injury causing ICA dissection and the need for a high index of suspicion for pseudoaneurysm formation as the pathology can be rapidly progressive, fatal, and may present with variable symptoms.

2. Case presentation

A 26-year-old man with no previous medical history was transferred to our institution after sustaining a gunshot wound to the left suboccipital area. Computed tomography angiography (CTA) followed by digital subtraction angiography (DSA) demon-

Abbreviations: AP, anteroposterior; CAD, carotid artery dissection; CT, computed tomography; CTA, computed tomography angiography; DSA, digital subtraction angiography; ICA, internal carotid artery.

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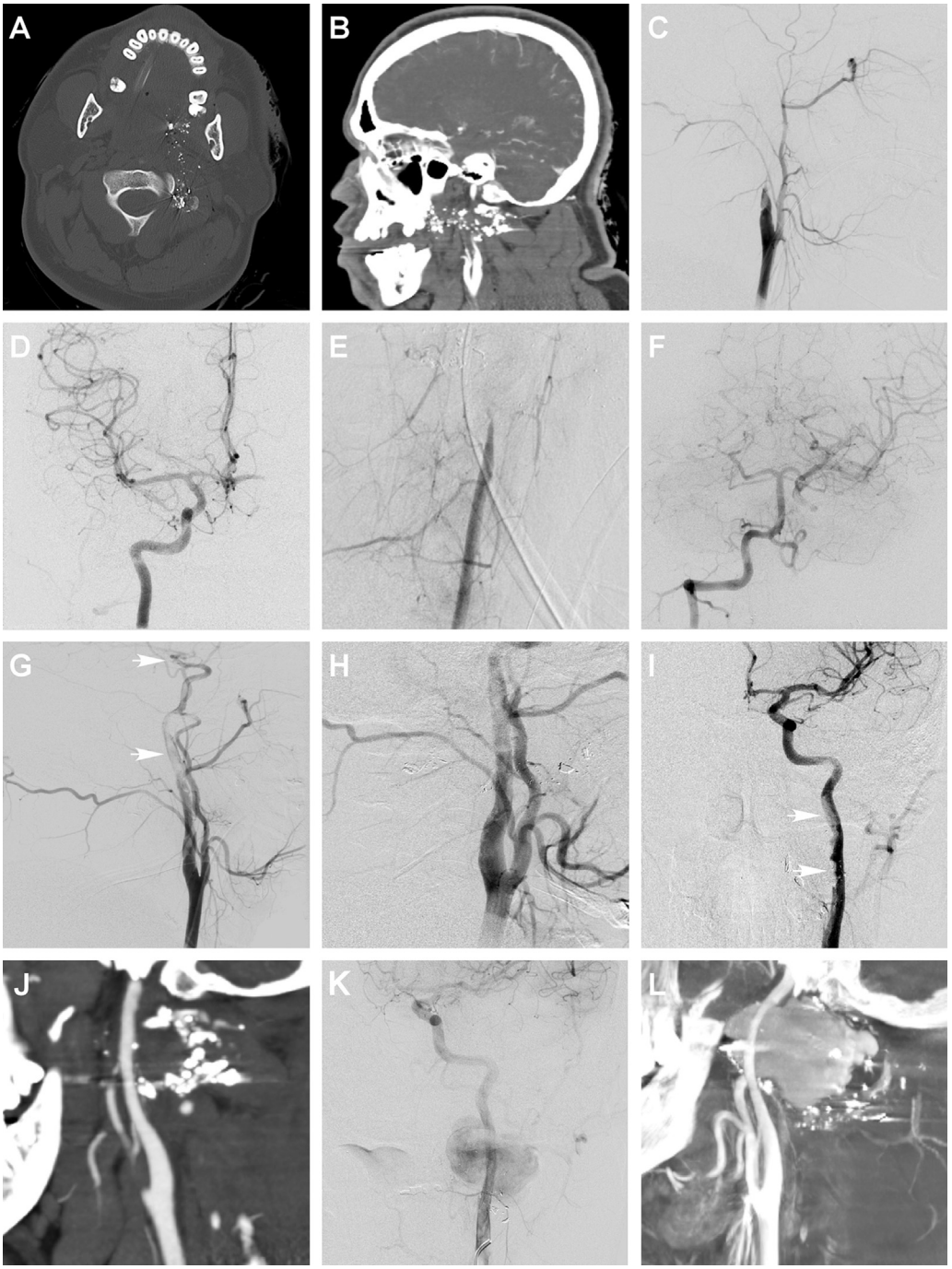

Fig. 1. (A and B) Initial presentation imaging. (A) Axial CT head demonstrating the gunshot wound and bullet fragments. (B) Maximum intensity projection CTA sagittal reconstruction demonstrating dissection of the ICA as well as bullet remnants filling the posterior oropharyngeal space. (C–F) Digital subtraction angiography (DSA). (C) Lateral left CCA injection demonstrating left ICA dissection with complete interruption of distal flow. (D) AP right ICA injection shows collateral circulation to the contralateral hemisphere. (E) AP left vertebral artery injection shows total occlusion at the level of C2. (F) Right vertebral artery injection demonstrates collateral circulation to the contralateral hemisphere. (G) Three days post-trauma, left CCA injection lateral view demonstrates partial recovery of flow in the left ICA with distal thrombus formation (arrows). (H) 8 days post-trauma, lateral left CCA injection, demonstrates total recovery of flow in the Left ICA. (I) AP projection of left ICA injection the same day demonstrates extensive thrombosis over the distal segment of the ICA at the supraclinoid level (arrows). (J) Maximum intensity projection CTA sagittal reconstruction 12 days later, after patient re-presented with hoarseness and dysphagia demonstrates continued recanalization of the dissected left ICA with no evidence of pseudoaneurysm formation. (K and L) Angiography six weeks post-trauma demonstrating a large, low-flow pseudoaneurysm. (K) AP left ICA DSA. (L) Left ICA injection cone-beam CTA maximum intensity projection sagittal reconstruction.

strated complete occlusion of the proximal cervical segment of the internal carotid artery (ICA) with a flame-shaped appearance suggestive of subintimal dissection of the vessel (Fig. 1C). It also showed supraclinoid left ICA filling defects consistent with intraluminal thrombus and complete occlusion of the left vertebral artery at the level of C2 vertebral body secondary to its fracture at the transverse foramen (Fig. 1A and E). Extensive metallic fragments could be seen along the left retromolar and parapharyngeal spaces (Fig. 1B). Collateral circulation from the right ICA via the anterior communicating artery and the right vertebral artery via the posterior communicating artery provided flow to the left cerebral hemisphere (Fig. 1D and F). Neurologic exam revealed mild left ptosis and right-sided dysmetria. The patient was otherwise neurologically intact. Otolaryngological consultation

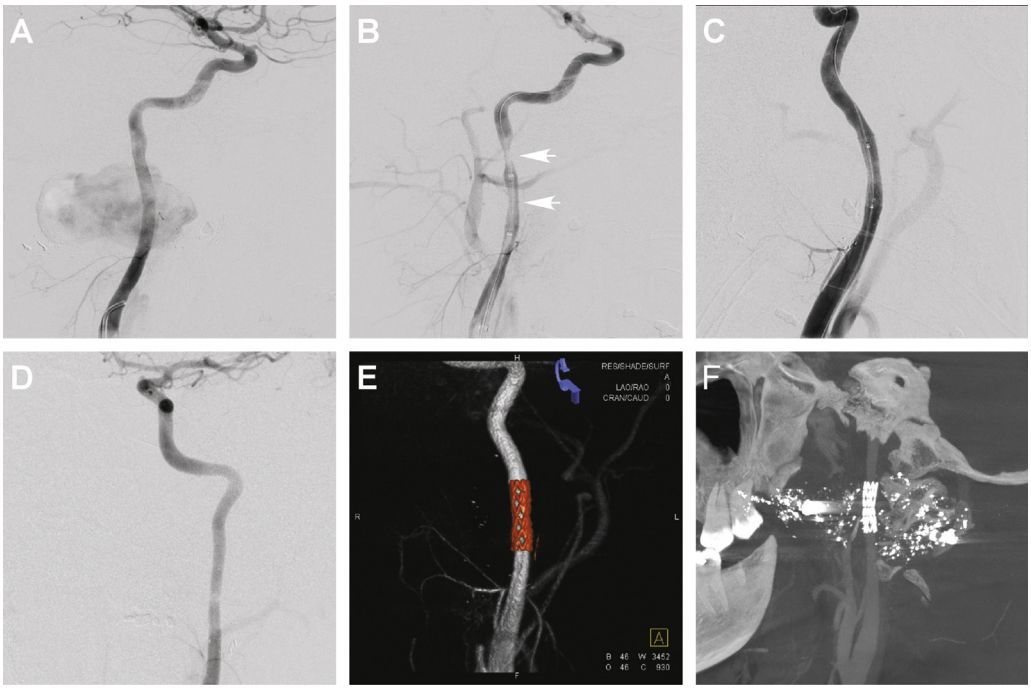

Fig. 2. Treatment and follow-up: (A) digital subtraction angiography, left ICA lateral projection demonstrating the large low-flow pseudoaneurysm and a stenotic region just distal to the pseudoaneurysm. (B) Left ICA lateral projection after stent graft deployment demonstrates no filling of the pseudoaneurysm but an area of mid-stent stenosis and vasospasm distal to the stent (white arrows). (C) Left ICA lateral projection after angioplasty. (D–F) Follow-up angiography 10 weeks later. (D) Left ICA AP projection demonstrating no residual pseudoaneurysm filling or vessel stenosis. (E) Three dimensional rotational angiography dual-density virtual projection and (F) maximum intensity projection, sagittal reconstruction cone-beam CTA demonstrating the stent graft without evidence of stenosis or malapposition.

was requested and a fibre optic laryngoscopy demonstrated mobile vocal folds bilaterally and no reports of neurological abnormalities. The patient was treated with intravenous heparin to prevent further thrombosis and was monitored in the intensive care unit. Repeat angiography imaging three and eight days later demonstrated progressive recanalization of the left ICA (Fig. 1G–J). The patient was subsequently discharged on subcutaneous enoxaparin (Lovenox, Sanofi, Bridgewater, NJ) ten days after admission.

Twelve days after the injury, the patient presented to the emergency department with hoarseness, night sweats, decreased sweating on the left side of the face, and dysphagia with liquids. Otolaryngological exam revealed immobility of the left true vocal fold. Examination of the neck and oral cavity was normal, without palpable mass or adenopathy. Gag was absent. Computed tomography demonstrated recanalization of the left ICA without evidence of pseudoaneurysm, and a small faintly enhancing fluid collection in the left semispinalis capitis. This was assessed as unlikely to be an abscess and the patient was discharged with plans for ongoing otolaryngological follow-up for vocal cord weakness. The vocal cord weakness and Horner syndrome remained stable over the next several weeks, and the otolaryngologist diagnosed a delayed cranial neuropathy with likely inflammatory aetiology.

Six weeks after initial presentation, a routinely scheduled follow-up diagnostic angiogram revealed a newly developed, 4 cm × 4 cm × 3 cm low-flow pseudoaneurysm of the left ICA (Fig. 1K and L). The patient was admitted to the neurosurgery service and stent-graft embolization of the pseudoaneurysm was planned. Under general anaesthesia, a balloon expandable 5 mm × 16 mm iCAST Covered Stent (Atrium Medical Corporation, Hudson, NH) was deployed covering the neck of the pseudoaneurysm and occluding flow into it (Fig. 2A and B). An Apex 5 mm × 15 mm over-the-wire balloon (Boston Scientific, Natick, MA) was used to angioplasty a mid-stent stenosis and the proximal and distal ends of the stent (Fig. 2C). Angiography five days then 10 weeks after the procedure showed no evidence of filling of the pseudoaneurysm, in-stent stenosis or branch vessel occlusion (Fig. 2D–F). The patient was maintained on dual anti-platelet therapy consisting of aspirin and clopidogrel (Plavix, Bristol-Myers Squibb Company, New York, NY). At 10 weeks follow-up, the patient reported significant improvement of the dysphagia and neck discomfort and modest improvement of the hoarseness.

3. Discussion

Little data regarding the natural history and time course of penetrating vascular injuries in the neck has been reported. While blunt injury to the neck has been associated up to 3% risk of dissection, penetrating injury causing dissection is quite rare.[5] Occult vascular injuries in the early post-traumatic course have let some authors to advocate for early re-evaluation and liberal use of angiography, especially in the military setting.[4] Additionally, development of chronic pseudoaneurysm years after penetrating trauma has been reported in the literature.[8] The subacute follow-up protocol of these lesions, however, has not been studied, and there are no guidelines for the optimal follow-up strategy of these lesions. On the other hand, blunt neck trauma causing dissection is more common and better understood. Biffl et al. reviewed 171 patients with blunt cerebrovascular injuries and found that follow-up angiography performed 7–10 days after injury demonstrated pseudoaneurysm formation in 8% of grade I and 43% of grade II injuries (intimal irregularities, and dissections with less than 25% luminal stenosis respectively) and resulted in a change in management in a majority of cases.[1] However, in patients with total vessel occlusion (grade IV), 82% of cases were unchanged on follow-up. Based on this observation, these authors recommended follow-up (7–10 day) imaging in grade I or II lesions in blunt cerebrovascular injury. We could not identify a similar report for penetrating injuries, but would suggest the risk of delayed injury may be even greater in this population since a higher degree of direct injury may be sustained from bullet cavitation, direct injury,

or fragments of bone. In the case discussed here, the patient presented with total occlusion (grade IV) and demonstrated early recanalization within the first week. Despite this, he went on to develop a large pseudoaneurysm sometime between 12 days and 6 weeks after injury. This phenomenon may be explained by exposure of the weakened vessel wall to increasing pressure as recanalization progressed. The time course of recanalization after complete occlusion, with or without medical therapy is not well understood, and has been reported to be up to 62% by 6 months in spontaneous dissection.[10]

Given the unclear time course of these lesions, a high clinical suspicion is warranted to help guide angiographic follow-up. Classically, CAD presents with head and neck pain, Horner's syndrome and cerebral ischaemia. Other reports in the literature point to pulsating tinnitus, transient monocular visual loss, ischaemic optic neuropathy and retinal infarct as unusual presentations of the disease, rarely occurring as isolated entities.[2] Cranial nerve palsy is a frequent manifestation of CAD and is present in 8–16% of cases.[7] The most commonly encountered palsy involves the hypoglossal nerve[3,11,6] and less frequently cranial nerves III, IV and VI. Cranial nerve palsy and Horner syndrome are thought to be from compression of the nerve by the expanding adventitial haematoma or pseudoaneurysm. In our patient, a paralyzed left vocal cord and loss of gag reflex may have been due to compression of the vagus and glossopharyngeal nerves by the expanding pseudoaneurysm. In the context of worsening dysphagia for liquids, an expanding pseudoaneurysm also compressing the pharynx was most likely.

Two important lessons can be learned from this. First, in patients with known vessel injury, a high degree of suspicion must be maintained for pseudoaneurysm formation. In this patient, despite re-presenting with dysphagia, hoarseness and a left vocal cord paralysis, CTA was negative for a new lesion and consequently a diagnosis of subacute inflammatory neuropathy was made, though this did not explain the patient's worsening dysphagia. The absence of contrast enhancement on CTA may have been due to the low-flow nature of the pseudoaneurysm, or possibly due to absence of flow in the false lumen during the contrast bolus. Secondly, we would recommend follow-up angiography within the first month to assess an injured vessel, especially in the setting of a changing pathology such as recanalization. The patient's dissection in this case had appeared to heal almost completely prior to discharge. A potentially fatal rupture from this pseudoaneurysm was prevented by timely re-imaging.

Taken together, we believe an ongoing high level of clinical suspicion combined with close angiographic follow-up may prevent morbidity and mortality from penetrating vascular injuries.

Conflict of interest statement

I Adel Malek, MD, PhD, certify that this manuscript is a unique submission and is not being considered for publication with any other source in any medium. The content of this manuscript has not been previously published elsewhere in any form. The senior author (A.M. Malek) has received unrestricted research funding from Codman Neurovascular (manufacturer of the Enterprise stent), Stryker Neurovascular (manufacturer of the Neuroform stent), Microvention-Terumo Inc., Siemens Medical Imaging, Ansys Inc., and CD-Adapco, and Boston Scientific Corp., for research that is unrelated to the submitted work. The other co-authors do not have any conflict of interest to declare with respect to the study. The research is original and was conducted by the authors with significant contribution from each.

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